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STUDIES ON BACTERIAL CANKER OF TOMATO

BY

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STUDIES ON BACTERIAL CANKER OF TOMATO [1]

By MARY K. BRYAN

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INTRODUCTION

Bacterial canker of tomatoes was first described by E. F. Smith (*24*)[2] in 1909. He cultured the causal organism, which he named *Aplanobacter michiganense*, proved its pathogenicity, and made some cultural studies of the organism and histological studies of diseased plants (*25*, *26*). He called the disease Grand Rapids disease of tomatoes, from the location where it was first found, but in his textbook (*26*) he renamed it bacterial canker of tomatoes. Doidge (*11*) in the same year used the name tomato canker for another disease occurring in South Africa. Gardner and Kendrick (*14*) have identified this South African disease with the bacterial spot of tomato and pepper occurring in the United States, caused by *Bacterium vesicatorium*; and the name "bacterial spot" used by them is commonly accepted for this disease in the United States. Kotte (*16*) suggests that bacterial wilt would be a better name than bacterial canker, since the cracks on the stems are not true cankers; but the existence of another bacterial wilt, caused by *Bact. solanacearum*, and the identification of a cankerlike fruit spot with the disease caused by *A. michiganense* make this name undesirable. It seems justifiable, therefore, to retain the name bacterial canker for the disease caused by *A. michiganense*.

A circular and several notes on bacterial canker have been published by the writer (*4*, *5*, *6*, *7*, *8*). The present paper gives in more detail the results of recent studies.

GEOGRAPHICAL DISTRIBUTION

Bacterial canker is a serious menace to both field and hothouse tomatoes. First described from Michigan in 1909, the disease was later reported from New York, Massachusetts, Pennsylvania, Connecticut, New Jersey, Ohio, and Illinois; but until 1927 it was not known outside of this northern group of States. A severe outbreak occurred in large commercial fields in New York in 1926 (*15*). In 1927 the disease was reported from Georgia, Utah,[3] and Montana; in 1928, from Washington (*21*, *p. 23*), California, and Wisconsin; and in 1929 it appeared in Indiana, Mississippi, and Maryland, and more

[1] Received for publication June 30, 1930; issued December, 1930.

[2] Reference is made by number (italic) to Literature Cited, p. 850.

[3] LINFORD, M. B. PLANT DISEASES IN UTAH IN 1927. U. S. Dept. Agr., Bur. Plant Indus. Plant Disease Rptr. Sup. 59: 100-101. 1928. [Mimeographed.]

widely than before in California (*20*). Since the fruit spot caused by *Aplanobacter michiganense* was observed as early as 1917 but was not identified with this disease and was later confused with the bacterial spot caused by *Bacterium vesicatorium*, the distribution is probably wider than here recorded. Certainly Iowa must be included on the evidence of a photograph by Pritchard (unpublished) of typical spotted fruit from Muscatine, Iowa, in 1917.

Bacterial canker has been reported from several foreign countries. Peglion (*22*) in 1915 reported a disease occurring in Italy which he considered identical with bacterial canker. McLarty (*18, 19*) noted its occurrence in British Columbia in 1925 and 1926. In 1928 Bisby and Conners (*2*) found it in Manitoba. Its first appearance in Germany has just been reported by Kotte (*16*). A disease said to resemble this disease more than the one caused by *Bacterium solanacearum* was reported as occurring in an Australian greenhouse (*23*) in 1925, but no cultures were made. The statement that the plants recovered with the onset of warmer weather makes its identity doubtful.

ECONOMIC IMPORTANCE

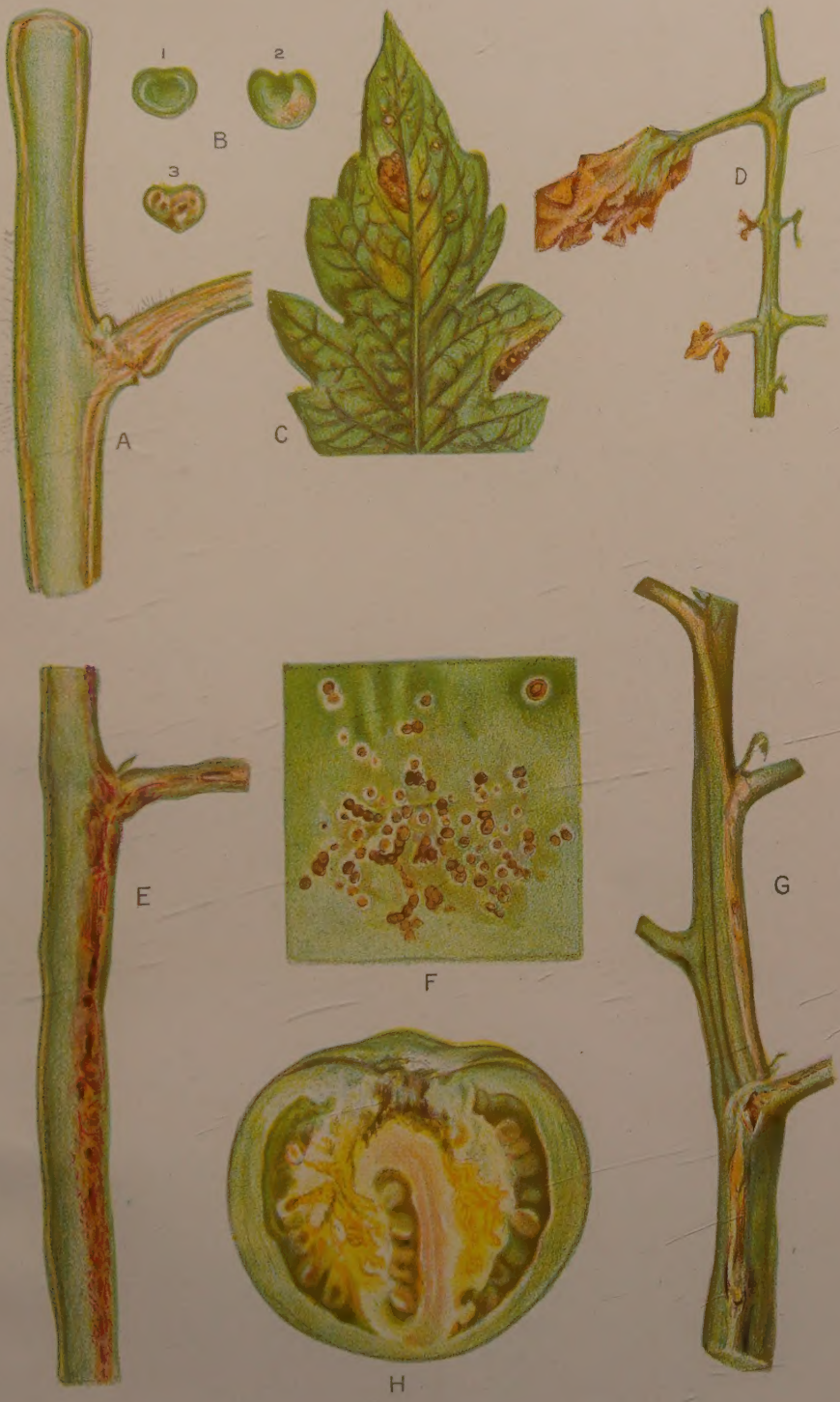
The widespread occurrence of bacterial canker and the fact that it is seed borne and is able to overwinter in the soil under some conditions, together with its very destructive nature, make this one of the most serious tomato diseases known. Field losses range from a trace to 100 per cent in individual fields. In many cases at the time when the crop should be in bearing there are very few living plants left in a field. (Fig. 1.) Such destructive outbreaks have occurred in Michigan, New York, Georgia, and Utah in large commercial plantings, and in smaller acreages in many other places. Fruit spot has caused losses of 25 to 75 per cent of the otherwise marketable crop in some fields in Georgia and Mississippi.

HOSTS

In the field the disease has been found only on tomato, although Smith (*25*) reported its spread to a spiny Porto Rican weed, *Solanum mammosum*, in the greenhouse. Repeated inoculations have failed to produce any infection on potatoes, eggplants, or peppers of either sweet or Cayenne varieties, or on the common solanaceous weeds *Physalis* sp. and *Datura stramonium*. On the other hand, no variety of tomato tested has been found resistant. The varieties tested include Marglobe, Marvelosa, Norton, Earliana, John Baer, Greater Baltimore, Bonny Best, Chalks Jewel, Stone, Globe, Red Pear, Walter Richards, Gulf State Market, Crystal Springs Market,

EXPLANATORY LEGEND FOR PLATE 1

A.—Longitudinal section of an infected stem. Early stage showing the yellow or cream-colored infected areas. Note the extensive invasion of the base of the petiole.
B.—Cross sections of bases of petioles: 1, normal; 2, early infected; 3, late stage of infection, with cavities.
C.—Leaflet showing leaf spots in several stages.
D.—Portion of leaf showing a streak on the rachis running out on a petiolule. Leaflets on the opposite side were healthy.
E.—Section of an infected stem at a later stage. Extensive invasion of the pith with cavities and darker color.
F.—Bird's-eye spots on green fruit.
G.—Streaks on a stem, running out onto petioles and beginning to crack open.
H.—Systemically infected fruit. Bacteria have flooded out from the vascular strands, making them appear broader than when freshly cut.



L. C. C. Krieger and J. F. Brewer, Fec.

(For explanatory legend see opposite page)

Ignotum, and a variety of the potato-leaf type. Kotte (*16*) reports Tuckswood, Triumph, and Lucullus as susceptible varieties.

PHASES OF THE DISEASE

Bacterial canker has two serious phases. The primary phase is a systemic infection which causes the gradual wilting and death of the plant and infection of the seed through the vascular system; and the secondary phase is a fruit spot which, although superficial, renders unsightly a large percentage of otherwise marketable fruit.

SYSTEMIC INFECTION

Effect on the Plant

The plant may go down at any stage, from seedling to the mature plant with a set of fruit. The disease usually progresses rapidly, but

FIGURE 1.—Tomato field in Georgia; vacant places caused by bacterial canker

several cases of recovery from early symptoms have been observed. Plants in the field may die before branching or setting fruit. Plants that have attained branching size show decided stunting of one or more branches, often with a thickening of the shortened internodes. (Fig. 2.) Light-colored streaks, sometimes becoming dark, appear on the stems, extending along one or more internodes or up the lower side of petioles or peduncles. (Pl. 1, G; figs. 3 and 4.) Narrower streaks also appear on the upper side of the rachis (pl. 1, D), and on the midribs and veins of the leaflets. These streaks, which follow the line of heavy infection in the cortex, crack open at intervals, forming cankers. (Figs. 3, 5, and 6.) The cankers on midribs and veins, though inconspicuous, are very common and from field evidence appear to cause more secondary infections than do the larger cankers on the stems.

One of the earliest symptoms, more noticeable on staked than on unstaked plants, is a turning downward of the lower leaves, accompanied by curling of the leaflets. An irregular wilting and

shriveling of the leaflets follows. When, as is very common, the infection runs on one side only of the petiole and midrib, a distortion of the leaf results, the leaflets on one side remaining healthy, while those on the other side die. (Fig. 3.) The petioles do not wilt while the leaflets are wilting and shriveling, but they may turn downward. The topmost leaves remain turgid until infection reaches

FIGURE 2.—Tomato plant diseased with bacterial canker, showing stunting and thickening of stem and irregular wilting

them, although in hot, dry weather wilting of the top has been observed; and at times, in the case of very severe infection, a general wilting of the plant occurs when only the lowest leaves show other symptoms.

In Mississippi, following extremely rapid, succulent growth due to abundant rain after drought, the first symptom on the leaflets was the appearance of sharply outlined pale-green spots of collapsed tissue lying between veins, often following the midrib. These areas, which sometimes were more than 1 centimeter wide by several centimeters long, later became gray as the leaflets wilted.

During the very wet weather occurring in Georgia in May, 1928, unusual symptoms were observed in many of the very succulent plants that were succumbing to the disease. Externally the younger parts of infected stems had a clear, water-soaked appearance; and

FIGURE 3.—Leaf showing infection on one side only. Streaks and cankers on the rachis and the midrib of the terminal leaflet

these, as well as adjacent infected petioles, were water-soaked inside instead of being mealy as infected tissues usually are. They showed the typical yellow color. This was the only time that such a condition was observed.

When a diseased stem is cut lengthwise the path of the disease is found to be very clearly marked by a line of creamy white, yellow,

FIGURE 4.—Streaks on stems and petioles. Note the bulging of the stem in A and irregular surface in B over badly disintegrated tissues

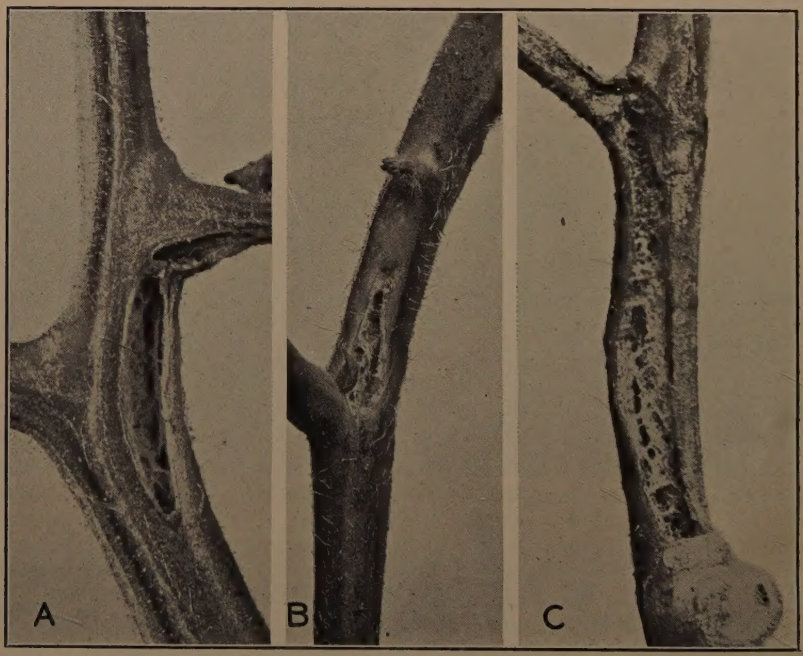


FIGURE 5.—Bacterial cankers on tomatoes: A, Canker on rachis, running out on a petiolule; B and C, cankers on stems

or reddish brown, just inside the woody cylinder (pl. 1, A and E), the reddish-brown color appearing in advanced stages. Along this line the pith cylinder is easily separated from the xylem. The discoloration is most conspicuous in the upper part of the plant where the bacteria have spread from the inner vessels of the wood to the more succulent phloem, pith, and cortex. Cavities of greater or less extent are formed in these areas. (Fig. 7.) The pith is often completely disorganized. The base of the petiole is especially susceptible and a cross section taken at this point is the easiest method of diagnosing the disease. Creamy white to yellow, mealy-looking areas in the pith or cortex, sometimes with cavities and in late stages with the red-brown tinge (pl. 1, A, B, and E), are clear evidence of bacterial canker.

FIGURE 6.—Old cankered stem and a stunted fruit on a cankered peduncle

Sometimes the root, especially the pith, is completely invaded; more often it shows very slight symptoms easily overlooked by the naked eye, but visible under a hand lens even when infection has taken place from infected soil through the roots; or it may be entirely free from infection. A slightly discolored line in the pith adjacent to the xylem is often the only sign of infection.

EFFECT ON THE FRUIT

Infection passes from the stem into the fruit through the vascular system and may be traced by the yellow infected veins directly to the seeds. (Pl. 1, H.) The infection spreads also into the tissues of the placenta and the fleshy covering of the seeds. In the hothouse overripe, heavily infected fruit has occasionally been observed of which the whole interior was disorganized and liquefied while the skin remained intact. Poured plates made from material removed aseptically from the interior of such a fruit gave pure cultures of *Aplanobacter michiganense*. Fruits infected when very young are

stunted and distorted. (Fig. 8, A.) Fruit that develops to nearly or quite normal size shows no external sign of internal infection. Yellow color is observable, however, in the scar made by removing the calyx. Heavy infection of the pedicel just beneath the calyx causes browning and shriveling of the sepals and loosens the attachment of the fruit. The tissues at the junction of pedicel and peduncle are very susceptible and frequently become so disintegrated that the fruit falls when the vine is shaken. Open cankers are common at this joint. (Fig. 8, B.)

In fruits of normal size the seeds do not show external evidence of internal infection, even when there is very heavy infection in the placenta and when the bacteria may be traced by the yellow veins directly to the seeds. Only in a stunted, distorted fruit, infected

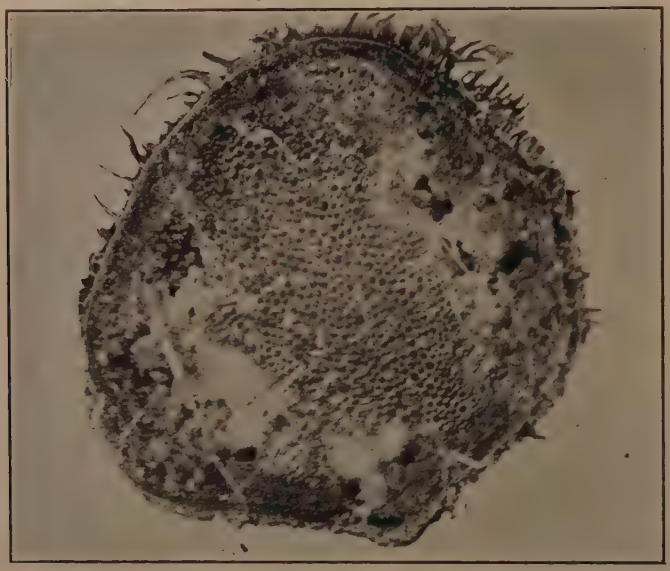


FIGURE 7.—Cross section of a diseased stem showing infected areas (white) and cavities forming in pith, xylem, and cortex. × 5

when very young, are the seeds abnormal. Then they are small, often black, and do not mature. A few such blackened or black-spotted seeds may be found in the larger fruits. Normal-looking seeds and discolored but normal-sized seeds from infected fruit give a high percentage of germination.

FRUIT SPOT

FIELD OBSERVATIONS

Fruit spot of the tomato was first observed by the writer in Georgia in 1928. Previous field studies had been made during the very dry season of 1927. In 1927 systemic infection was very severe in Georgia, but no fruit spot was noted. In the same location the following year systemic infection was again very serious. This was an unusually rainy season, and the idea that drought was a factor in the severity of the disease had to be abandoned, since the systemic infection was as severe as in the preceding dry year. On the first set

of fruit small, round white spots, some with brown centers, were noticed. Specimens were brought to Washington and isolations made. *Aplanobacter michiganense* was obtained in every case. Later the fruit spotting was so widespread in these fields that a large percentage of otherwise salable fruit had to be discarded. The next year (1929) there was a much smaller percentage of both the systemic disease and the fruit spot in these fields.

In 1929 bacterial canker was observed for the first time in Mississippi. (Fig. 9, A.) Fruit spot was more conspicuous than the systemic infection in these fields; that is, there were very few vacant places, but 75 per cent of the fruit that had set was spotted. There had been unusually heavy wind and rain storms, and the large number of

FIGURE 8.—Systemically infected fruits. A, Stunted, distorted fruit; B, infected fruit spur. Note cankers at junction of pedicels and peduncle, which cause the fruit to fall readily

fruits that were spotted on the side toward the prevailing wind gave good circumstantial evidence of spread by wind-blown rain. Open cankers on leaves or petioles could usually be found on the plants in the next row. This was especially significant when there were no cankers on the plant bearing the spotted fruit.

Good evidence that infection was carried from the leaf cankers to the fruit in the drip from rain or dew was obtained in Maryland and New Jersey, where only a small amount of disease was found and where the more conspicuous fruit spots led to the finding of the small leaf cankers just above. Fruit spot has also been traced to infection coming from open cankers on sepals or at the junction of fruit and calyx. Here where moisture is held by the calyx, providing ideal conditions for infection, elongated or irregular brown spots with white margins form along the edge of the scar. In some cases white

areas several millimeters in extent were observed in which a central breakdown had not begun. Groups of typical bird's-eye spots on the

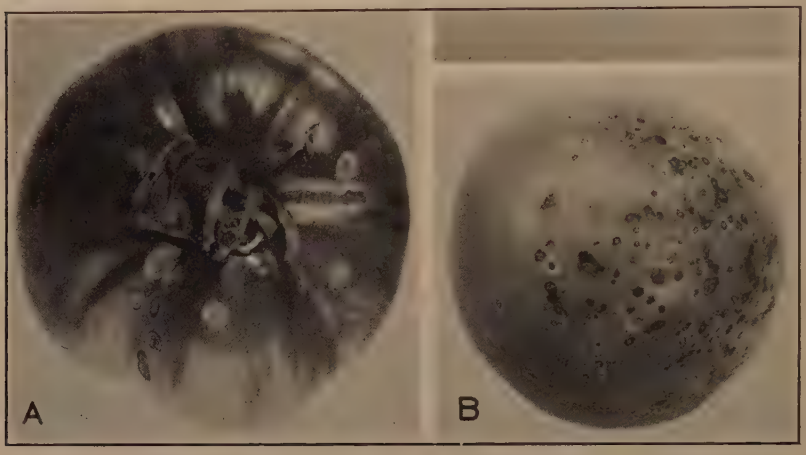


FIGURE 9.—A, Infected sepals, canker with white border running down from point of attachment, and large bird's-eye spots; B, numerous small bird's-eye spots

side of the fruit appear to have resulted from infection borne by drops of moisture from the open cankers under the calyx. (Fig. 9, B.)

FIGURE 10.—Bird's-eye spots on a young green fruit, produced by spraying with *Aplanobacter michiganense*, photographed 16 days after inoculation. × 2

The lesions on the fruit are at first minute, round, snowy white, slightly raised spots occurring singly or in large or small groups. (Fig. 11, A.) As these develop their centers become pustular and

break open with a single rift or with many small cracks, showing a roughened yellow-to-brown surface. (Fig. 10, Fig. 11, B, and Pl. 1, F.) The white persists indefinitely as a conspicuous halo, although in some old spots it disappears partially or completely. Spots may be so numerous as to form large, crusty patches. There is no water-soaking at any stage of development. The spots remain small, not exceeding 3 mm. in width, exclusive of the halo.

Isolations and Inoculations

Bacteria were repeatedly isolated from the spots with white halos. Isolations are most easily obtained from fruit spot by swabbing the surface repeatedly with sterile water, removing a group of spots with a sterile knife, and crushing them in a tube of beef broth from which the plates are then poured. Slant agar transfers from the resultant colonies that appeared typical for *Aplanobacter michiganense* were used to make prick inoculations on stems of healthy tomato plants in the hothouse. Typical systemic infection was obtained, the organism exhibiting as much virulence as freshly isolated, authentic isolations of *A. michiganense*. With the same culture fruit spot was produced by lightly swabbing young fruits from one-half to 1 inch in diameter with water suspensions from agar slants, using a small wad of cotton. Later, fruit infection was obtained by spray inoculations with the same isolations. (Figs. 10 and 11.) Reciprocally, fruit spot was readily produced by inoculation with *A. michiganense* isolated from systemically infected stems. White spots one-half millimeter wide were usually noticed from four to six days after inoculation by either swab or spray. The size of the subsequent spot and the rapidity of the development of the roughened center depend on the age of the fruit. The largest spots developed on fruits that were very small (one-half to 1 inch in diameter) when inoculated.

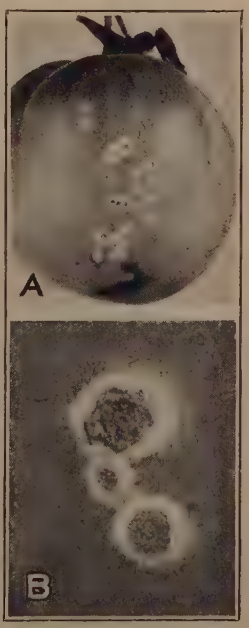


FIGURE 11.—Spots produced by spray inoculation. A, Very young spots five days after inoculation. × 5. B, Enlargement of portion of Figure 10 to show the rough, cankered centers of the spots. × 6

Comparative inoculations were made on young fruits with a culture of *Bacterium vesicatorium* received from Gardner. The resultant spots were always like those described by him. Water-soaking was always present, and no spots with white halos appeared.

Histology

Gardner and Kendrick (*14*) stated that since there are no stomata on tomato fruits and typical infection with *Bacterium vesicatorium* was not obtained by means of insect punctures, bacteria probably penetrate the epidermis through minute rifts in the cuticle or through broken hairs. In this they are doubtless correct. The young fruit is densely covered with two types of very delicate hairs, one a long slender trichome, the other a short hair with a several-celled glan-

dular head. Sections of fruit with young bacterial canker spots have shown the bacteria in the bases of hairs (fig. 12, A) and in the intercellular spaces of the adjoining subepidermal layers.

Fruit spots remain very superficial, not penetrating as deeply as those caused by *Bacterium vesicatorium*, but otherwise much like

FIGURE 12.—Sections of spots on green fruits. A, Bacteria in the base of an epidermal hair and in the subepidermal tissues. × 40. B, Section through a young spot. × 65

them in development. (Fig. 12, B.) The cells below the infected part multiply noticeably in an attempt to cut it off with a cork layer. There is also a multiplication of subepidermal cells in the region of the white halo, but the white appears to be partly due to the presence of air in the tissues, since it becomes inconspicuous when the specimen is put into alcohol.

Earlier Observations on Bird's-Eye Spot[4]

In 1918 Coons and Nelson (*10*) published a brief note on a tomato fruit spot which they called canker, found in several widely separated regions. They described the spots as white, and the single illustration tallies with the fruit spot caused by *Aplanobacter michiganense*. Photographs and notes kindly furnished the writer by Nelson show that this was the predominant type of spot. Coons (*9*) in a later paper says: "The disease is disfiguring enough to cause serious loss to the crop." His illustration in this case, however, looks like the spot caused by *Bacterium vesicatorium*. Coons and Nelson isolated yellow organisms from both types of spot, but inoculations were made only with the one from dark spots. Nelson's photographs of inoculated fruit give clear evidence that they were dealing with *Bact. vesicatorium*. Their cultures were lost before Gardner and Kendrick studied *Bact. vesicatorium*, so a comparison of the organisms could not be made.

Gardner and Kendrick (*12*, *13*, *14*) also found the white spots, which they called "bird's-eye spot." Their illustrations of this type of spot are absolutely typical of bacterial canker. The legend accompanying the illustration states (*14*): "Bird's-eye spot of tomato. Cause not proved. Possibly a form of bacterial spot." They did not succeed in isolating an organism from these bird's-eye spots, nor did they produce them by inoculation with *Bacterium vesicatorium*. They considered that they were working with the disease described by Coons and Nelson, as they say (*13*): "The disease as it occurs on tomato fruit has been known for several years. Coons and Nelson, and later Coons, briefly describe this disease as tomato canker." It is interesting that the spot originally called canker proves to be a phase of the disease now bearing that name.

McCubbin (*17*) in 1918 figures early and late stages of what he calls black scab and which Gardner and Kendrick (*13*) say appears to be bacterial spot. The figures of the early stage, however, show only white spots, which are typical of bacterial canker spots in the early stage. It would seem, therefore, that McCubbin had both diseases under observation.

SECONDARY INFECTION

Where fruit spot is present in the field it is usually accompanied by spotting of peduncles, pedicels, calyx, and young stems. This takes the form of small, tan-colored, open cankers from 1 to 2 mm. wide. These remain very superficial, and although they occasionally have been found to penetrate deeper and reach the vascular system on inoculated plants, progress is so slow as to be of no practical importance. Leaf spot has not been certainly identified in the field, because of the abundance of fungus spots that confuse the picture. The only place in which leaf spot has been observed to occur naturally and has proved to be caused by *Aplanobacter michiganense* was an abandoned "spotting" bed in Georgia, where the overgrown plants, many with open cankers, were in a tangled mass. (Fig. 13.)

Spotting on stems and leaves has been obtained in the greenhouse by spray inoculations. (Fig. 14.) Infection appears to start fre-

[4] The name bird's-eye spot, used by Gardner and Kendrick for this spot, the cause of which they did not determine, should be retained because of its descriptive value.

quently in the large bulbous bases of the giant hairs. Stomatal infection was recorded and figured by Smith (*26*). Leaf spots are minute and extremely inconspicuous, creamy white, and raised by the mass of bacteria in the tissues. These may remain indefinitely in this condition or dark rings of dead tissue may form around individual spots (fig. 14, A and B, and pl. 1, C) and merge into larger dead areas (fig. 14, C). Occasionally infection reaches the vascular system from such spots, especially from those lying over the very large bundle of vessels on the extreme leaf margin. Where such spread occurs the leaf blade yellows from the margin inward and the veins blacken. (Fig. 14, D.)

SPREAD IN THE FIELD

No positive evidence of field spread of systemic infection by any agency has been obtained. The greenhouse spread recorded by

FIGURE 13.—Naturally infected leaves from an old spotting bed in Georgia. *Aplanobacter michiganense* was isolated from spots on these leaves

Smith has never recurred in the writer's experience. Moreover, the plants throughout the field in many cases succumb at approximately the same time. Slight variations are readily accounted for by the varying times at which infection takes place during the weeks in the seed bed. Spread in the greenhouse by the pruning knife has been reported by Coons (*9*) and by Williams (*28*). The disease has not been observed in fields where pruning was practiced, except in Mississippi; and there no convincing cases of spread were observed. Pruning in the field is often done by twisting out the side shoots. This, while not carrying the disease from plant to plant on the knife, leaves wounds for the possible entry of the bacteria. However, the disease progresses downward in the plant very slowly. The bacteria have been traced down from leaf inoculations through the petiole into the stem, but the spread from there is upward. When inoculation is made directly into the stem the spread downward is slow. Kotte (*16*) has recorded the same observation.

FIGURE 14.—Leaves inoculated by spraying with *Aplanobacter michiganense*. A, Upper surface of leaflet five days after spraying. Natural size. B, Leaflet dying from systemic infection; note blackened veins. × 2. C, Enlargement of a dead area in which small individual spots are distinguishable. × 2. D, Leaflets dying back from marginal infections. Natural size

INTERNAL INFECTION OF SEED

The presence of the bacteria inside the seed coat has been demonstrated by microtome sections and by cultural methods.

Sections of infected seed show that the bacteria lie in the fleshy outer seed coat (fig. 15), which is removed in the seed-cleaning process, and between the layers of the hard seed coat, penetrating the entire circumference of the seed in cases of severe infection. (Figs. 16, 17, and 18.)

Plates were poured from seed taken from fruits that showed infection in the placenta. Since not all seeds are internally infected, 10 or more were used for a culture. The seeds, after the removal of the fleshy, outer seed coat, were soaked in mercuric chloride, 1 to 1,000, for five minutes, rinsed thoroughly in sterile water, and crushed in beef broth. Plates were then poured and *Aplanobacter michiganense* obtained in pure culture. Isolations, identified by inoculations on tomato, have been made from seed 2½ years old.

FIGURE 15.—Section of seed from which the fleshy covering had not been removed, cut through the hilum at right angles to the flattened surface and parallel to the long axis. Bacteria are abundant in the dark areas of the seed coat beneath the hilum and running up both sides of the seed. The fleshy, outer seed coat is heavily infected. × about 40

MEANS OF DISSEMINATION

Infected seed, and plants grown from infected seed or in infected soil and shipped to other localities, are the means of spreading bacterial canker from place to place. There is also evidence of its having been carried in manure. Seeds are infected both externally and internally. The heavy infection in the placenta of diseased fruits insures wholesale contact of bacteria with the seeds in the process of seed saving. The bacteria are able to survive in a virulent condition eight months or more on the seed or on cover glasses and two and one-half years in dry soil.

EVIDENCE OF DISEASE FROM INFECTED SEED

That disease results from planting infected seed is attested by wide field observations and by experimental planting of seed from infected fruit. From 1 to 5 per cent of infection has resulted in the greenhouse from naturally infected seed and from 21 to 40 per cent from seed dipped in bacterial suspensions, dried, and planted. Field tests in Georgia with seed saved from infected fruit gave 53.4 per cent of cankered plants in 1928 and 30 per cent in 1929;[5] checks grown from seed known to be clean gave only healthy plants.

[5] Communication by letter from O. C. Boyd.

A wide discrepancy has been observed in the percentage of diseased plants obtained from seed known to be infected but grown at different times and in different places. For example, seed known to be infected, which gave only 1 per cent of infected plants in the greenhouse in Washington, gave 54.4 per cent in Georgia. This indicates that seed-bed conditions are an important factor in the production of devastating outbreaks of bacterial canker. Since tomatoes are not grown to maturity commercially in the tomato-plant growing region of Georgia and reliable plant growers rotate their fields or use newly cleared pinelands, soil infection does not appear to be a factor in producing diseased plants in this region. To determine this point clean seed was planted in 1930 in two fields in Georgia from which infected plants were shipped in 1929. No bacterial canker developed on the resulting plants, part of which were grown to maturity in the seed field and part of which (400 plants) were set out in Maryland.

FIGURE 16.—Section of a seed parallel to the flattened sides, showing bacteria (dark areas) in the tissues beneath the hilum and completely encircling the seed. × about 25

SPREAD IN THE SEED BED

A few primary infections under crowded seed-bed conditions are potentially able to increase the number of diseased plants to 100 per cent, if conditions are favorable. Such spread was found in an old, abandoned plant bed in Georgia during a very rainy season. The plants, which were beginning to set fruit, were in a tangled mass, so that spread was facilitated. Spots were abundant on fruit, stem, and leaf. In many of the plants examined no internal lesions were found in the root or lower stem, although in the tender upper stem they were conspicuous, indicating that systemic infection had resulted through primary lesions on upper stems or leaves. Plants are set in the field or shipped, however, while quite young, and spotting has not been found at this stage. Moreover, severe outbreaks in the field have occurred from hothouse-grown plants where weather conditions do not enter as a factor. In several cases all diseased plants could be traced to one hothouse or coldframe. It would seem that

there must be some other cause of spread, possibly insects, in the seed beds. A study of this problem is under way.

SEED TREATMENT

For externally contaminated seed, mercuric-chloride treatment has been found most effective. Such treatment, however, does not control internal seed infection, which is the more serious phase of the disease. For the internal type of infection hot-water treatments were undertaken. A study was first made of the thermal death point of the causal organism at various ages.

THERMAL DEATH POINT OF THE CAUSAL ORGANISM

The thermal death point of *Aplanobacter michiganense* has been found to vary in different strains from 50° to 53° C. This, however, refers to young 24 to 48 hour old cultures in thin suspensions in beef broth. To ascertain whether it would hold for older cultures or for bacteria massed on the seed and dried there, the following tests were made:

FIGURE 17.—Enlargement of a portion of Figure 16 to show the position of the bacteria: *a*, Caulicle; *b*, endosperm; *c*, inner wall of seed coat; *d*, layer of bacteria; *e*, outer roughened wall of the seed coat. × 170

1. Seeds were sterilized in the oven to eliminate saprophytes and thus facilitate the reisolation of *Aplanobacter michiganense*, which is a slow grower. These seeds were then immersed in a suspension from 10-day agar slants of *A. michiganense*, drained, and dried. They were then dropped into sterile water in test tubes and held in a water bath at 55° and 56° C. for 10 minutes and 20 minutes, after first being warmed for 5 minutes at 45°. The bacteria were cultured from these seeds in every case.

2. Bits of the surface agar of slant cultures 20 days old were dropped into beef broth and exposed for various time-temperature periods. Typical bacterial growth in transfers made from these cultures after exposure demonstrated that the bacteria in this condition are not killed by exposures of 20, 30, or 40 minutes at 54° C. or by an exposure of 10 minutes at 55°.

HOT-WATER TREATMENT OF SEED

Two sets of hot-water treatments of seed were carried on, the first to determine the effect on the seed, the second to ascertain the point at which the bacteria are killed.

1. Freshly cleaned, healthy seeds were tested to determine the point at which injury occurs. The seeds, covered with water in test

tubes, were first exposed in a water bath at 45° C. for 5 minutes, then plunged into water at 50°, 52°, and 54° and kept at these temperatures for 10, 20, 30, 40, 50, and 60 minutes. Germinations were counted in 100 seeds of each lot placed between damp blotters. One hundred seeds of each lot and 100 check seeds were planted in the greenhouse and watched for sturdiness. Although there was slight retardation after the longer time treatments at 54° (3 days at 54° for 40 to 60 minutes), there was no reduction in percentage of germination or vigor of plants in any case. The 50° treatments stimulated earlier germination than took place in the checks. Plants were potted and held until they had reached a height of 8 inches. The same results were obtained with dry seed treated at these temperatures. Similar experiments at 55° and 56°, for 30 minutes and over, produced decided injury to wet, freshly cleaned seeds, both in germination and vigor of plants. Dry seeds were not noticeably injured when treated for 30 minutes at these temperatures but when exposed for longer periods gave a decidedly lower percentage of germination and weaker plants.

FIGURE 18.—Section of a seed cut in the same plane as Figure 16 and at a point corresponding to X in Figure 16, showing bacteria massed in the vascular region and more scattered in the adjacent loose tissue. × 210

2. Seeds from infected fruit were treated with hot water for the various time-temperature periods used in experiments with clean seeds. After such treatment mercuric chloride, 1 to 1 000, was used for 2 minutes to eliminate any saprophytes that might be present on the surface. The seeds were then rinsed in sterile water and crushed fine in beef broth. Since not all seeds are internally infected, 10 or more seeds were used for isolation purposes. The bouillon with the crushed seeds was poured into test tubes containing potato cylinders. Untreated diseased seeds were cultured in the same way, and the resultant yellow bacterial growth tested for its identity. In this way *Aplanobacter michiganense* was cultured from the checks and from seeds treated at 53° and 54° C. for 30 minutes, but not from those treated for 40 minutes. Since 55° is injurious and since commercially it is difficult to hold the water bath at a given temperature without variations of more than a degree, this does not

allow a sufficient margin of safety for general use. Further investigation may show that the method, although impracticable for commercial seed treatment, might be used in cleaning up a seed stock. The only real remedy appears to be in growing clean seed. The only way to obtain clean seed is to select from fields in which there is no bacterial canker.

PERSISTENCE OF BACTERIA IN THE SOIL

Laboratory tests to determine conditions under which the organism can live in the soil have shown tolerance of a wide range of temperature, moisture, and pH values.

Tubes of soil were sterilized, then inoculated with water suspensions of *Aplanobacter michiganense* sufficient to moisten. Half were sealed with paraffin to retain the moisture, the other half were left unsealed and soon dried. Part were held out of doors protected from rain in Washington and part at Geneva, N. Y., from June 15 to April 1. At the end of this period the organism was recovered by cultural methods in virulent form from both wet and dry soil from both places. In another experiment the organism survived two and one-half years in dry sterilized soil exposed to outdoor temperatures in Washington, D. C.

Preliminary experiments have been made in unsterilized soil sealed to retain the moisture and kept at low temperatures in refrigerators. These experiments have demonstrated that the organism will survive five months at 0° F. in soils from different sources with a pH of 5.67, 6.67, and 7.9, respectively. The viability of the organism was determined by inoculation, as it had been found impracticable to isolate *Aplanobacter michiganense* from unsterilized soil. For this purpose the soil culture to be tested was shaken with water for five minutes, allowed to settle, and the suspension thus obtained was used for prick inoculations on young tomato plants. Inoculations were also made by dipping the roots of very young plants in the suspension.

Tomato plants readily become infected when planted in freshly inoculated soil or when inoculum is poured into the soil around them in pots in the greenhouse. However, when plants so infected were allowed to die in the pots, the old vines removed, and young plants set in three months later, no disease resulted. This agrees with the report of Williams (*28*), who recorded inability to hold the organism in greenhouse soil for even a few months. An attempt to determine persistence in the soil in the open at Washington, D. C., was made. Twenty-four 8-inch pots filled with sterilized soil were buried to their tops in the ground in October. Leaves and stems of infected plants were buried in the upper layers of soil in 14 of the pots. In May the soil in the pots was worked. A water suspension of *Aplanobacter michiganense* was poured into the soil of 6 pots that had not been inoculated in the fall, and the remaining 4 were held as checks. Young Stone tomato plants were set in all of the 24 pots. The plants were watched all summer, but no disease developed in the checks or in the pots inoculated in the fall with diseased plants, although all of those in the freshly inoculated soil succumbed early to the disease. Since this one negative result is not conclusive, the experiment is being repeated, this time without pots.

Negative evidence was obtained in western New York, where for two successive years the disease did not appear in a field that had been a total loss from bacterial canker the preceding year. Further observations and experiments are needed before there can be any certainty in regard to this region.

There is positive evidence, however, that the disease can and does overwinter in the soil in some localities. In Georgia the study of soil hold-over was complicated by the bad field practice of the growers who used infected vines in the compost that was placed under the soil of spotting beds. By this practice almost 100 per cent of the plants became infected. When in 1929 the practice was abandoned there was only from 5 to 10 per cent field infection. This, while indicating hold-over in the soil of fields, is not so large a percentage as might have been expected in fields that had developed 50 to 75 per cent of infected plants the two preceding years. That the bacteria survived composting and were virulent in this compost the second year was demonstrated by Boyd.

Indication of a small percentage of disease from hold-over in the soil was observed in 1929 in California and Utah but was not conclusive because the seeds used were not known to be clean. In Illinois only one diseased plant was found in a half-acre field which had had 25 per cent infected plants the preceding year. Further observations are needed to determine the length of time the infection will remain in the soil. The small percentage of infection so far observed in fields infected the preceding year and the fact that the disease does not attack other crops or the weeds so far tested would indicate that rotation of crops should eliminate the bacteria from the soil. This theory is further supported by the fact that no bacterial canker could be found in 1930 in two fields in Georgia planted in tomatoes for the first time since 1927 when the infection was severe.

CONTROL

Control of bacterial canker is a difficult problem. Seed infection, soil infection, and spread in the seed bed must all be taken into consideration. Where the disease has not occurred, the use of clean seed is the first essential for safety. Seeds brought into disease-free areas for breeding purposes should be grown in separate seed beds, the trial plots carefully inspected, and any infected plants burned.

Seed should not be saved from fields in which even a small percentage of bacterial canker is found. Seed not positively known to come from clean fields should be treated with mercuric chloride, normal Semesan solution, or Semesan Jr. dust. This will eliminate surface infection and is desirable from the point of view of other tomato diseases, but no treatment is known which will destroy internal infection, under commercial conditions, without danger of injury to the seed.

Where the disease has occurred, rotation of crops also is essential. The number of years that the bacteria will live in the soil under field conditions when rotation is practicable has not yet been demonstrated. The small percentage of diseased plants on fields infected the preceding year, the absence of the disease from two old fields after two years in other crops and the fact that other crops and solanaceous weeds are not attacked make this a promising method. Where ro-

tation is impracticable, the development of a resistant strain of tomato would be desirable. The removal and destruction of diseased vines to reduce the amount of infection in the soil would also be desirable under these conditions. Kotte (*16*) suggests that infection through roots broken in transplanting may be prevented by dipping the roots in a 0.5 per cent solution of Uspulun thickened with soil to the consistency of a thin mud. A preliminary experiment was made in the greenhouse with Kotte's method, in which normal Semesan solution was used instead of Uspulun when setting young plants in heavily inoculated soil. This method resulted in a 20 per cent infection in the treated plants as compared with an 80 per cent infection in the checks.

Care should be taken in the sanitation of the seed bed. Williams (*28*) reports satisfactory control of the disease in greenhouses by sanitation but gives no details. No infected refuse, either vines or spotted fruit, should be returned to the soil or allowed to reach the compost heap or the barnyard. The soil of old beds in cold frames or hothouses should be replaced with clean soil from fields where tomatoes have not been grown, or it should be sterilized by steam. Hothouses should be fumigated. In the South, where seeds are sown in the open, fields to be used as seed beds should be burned over before plowing to destroy insects in plant refuse. Fall plowing would also aid in decreasing the insect population. A field should not be used two successive years for plant growing for the best results from several points of view.

Very few plants show any sign of infection at the time of setting in the field. Inspection for disease at this stage would be impracticable.

THE ORGANISM

VARIANTS

An albino strain has been isolated from single-colony cultures of *Aplanobacter michiganense* (*8*). So far as known, such a variant has not been reported for any other plant pathogene. Brown (*3*) found rough, rubbery, round white colonies on reisolation plates of *Bacterium beticolum* with which she obtained infections on sugar beets, but she did not record their cultural identity with that organism, nor did they appear in single-colony cultures of the typical yellow organism. White variants that do not revert to the typical color have been described by various workers for bacteria in other groups, notably *Bact. prodigiosus*, *Bact. violaceum*, *Staphylococcus aureus*, and several saprophytes from plants.

White streaks were observed in the surface growth on old agar stab cultures at three different times and with isolations from different places. On one occasion plates were poured from the white part of a culture of a strain isolated four years previously, and both white and yellow colonies appeared. These were alike except in color. Transfers from white colonies were found to be nonmotile and Gram-positive. No further work was done at that time and the cultures were lost. When in 1929 the same phenomenon occurred in a strain held two years on culture media, white colonies were again isolated. These were tested as before and also used for inoculating tomatoes. Prick inoculations were made in the stem and swab inoculations on young green fruits. In both cases typical lesions were produced,

the organism proving as virulent as the typical yellow form. Reisolations were made from petioles at intervals of 12 days, 6 weeks, and 2 months after inoculation, from near the point of inoculation and from the upper limits of the bacterial invasion. In all cases only white colonies appeared on the plates. In cultural tests on potato, litmus milk, Thaxter's potato agar, gelatin, beef broth, nitrate broth, and starch agar, the white form resembled the yellow form except in color. No yellow pigment was produced at any time.

Variants of another type were observed on whey agar and Thaxter's potato agar. On both of these agars containing sugar, on which the typical colony growth is abundant, fluid, and spreading, varying percentages of small, round, very convex colonies have appeared. These remain small and never spread. When replated on these agars the round type gives only round colonies; the spreading type may throw off a few round colonies. Replated on beef agar they are indistinguishable. The round colony has appeared in small numbers in young isolations but is much more common in isolations that have been on culture media for some time. In one 10-year-old strain only the round type was present. In this case no infections could be obtained. Round colonies of younger isolations are very weakly parasitic and weak in their reactions on culture media as compared with the spreading strain. They have not, however, lost the power of fermenting sugars.

LOSS OF VIRULENCE

Smith (*25*) has reported that *Aplanobacter michiganense* loses its virulence in the course of several years. Tests with various isolations—three of 1924, three of 1925, and one of 1926—were used in 1929 to make prick inoculations in the upper stems of large plants. All except two of the 1924 isolations showed symptoms of infection by the tenth day, and the disease developed rather slowly. Inoculation of the stems of young plants with the same cultures produced rapid and complete destruction of the plants, with typical symptoms. Virulence is therefore not lost by five years on culture media. As noted under "Variants," a 10-year-old culture failed to give any nfection.

MORPHOLOGY AND STAINING REACTION

Aplanobacter michiganense is a small, nonmotile rod, measuring 0.6 to 0.7 μ by 0.7 to 1.2 μ, and occurring singly or in pairs. It is definitely Gram-positive. No spores have been observed. It stains readily with the usual bacterial stains. Capsules occur in agar cultures.

CULTURAL CHARACTERS [6]

BEEF AGAR.—Growth in beef agar is slow, moderate, opaque, smooth, glistening, pale yellow becoming mustard yellow,[7] and not opalescent. Colonies on beef-agar plates may be seen under the hand lens on the second day but usually are not visible to the naked eye until the third day. They are then very minute, 1 mm. or less in diameter, very pale yellow, and very convex. By the fifth day they have reached a diameter of 2 to 3 mm., are round with an entire margin, smooth, shining, of butyrous consistency, opaque, pale yellow, and show no markings by either reflected or transmitted light. In 12 days colonies on thinly sown plates may attain a diameter of 6 mm. At this time they are bright yellow

[6] Unless otherwise stated, all beef media were made with beef infusion and had a pH value of 7.0 to 7.2.
[7] RIDGWAY, R. COLOR STANDARDS AND COLOR NOMENCLATURE. 43 p., illus. Washington, D. C.

and very viscid. Growth on the surface of agar stabs is round and restricted, attaining a width of 7 or 8 mm. by the eighth day. The stab growth is saccate and granular, visible to one-third the depth of the agar. On slant agar there is moderate growth, not running down into the V. On beef-extract agar growth is less abundant, paler (straw yellow) and thinner than on beef-infusion agar; it is opalescent and does not become viscid.

THAXTER'S POTATO AGAR.—On this medium growth is very abundant, fluid, long continued, and paler than on beef-infusion agar, though not so pale as on beef-extract agar. By the sixteenth day colonies may attain a diameter of 15 mm. and show no sign of drying down but remain thick and fluid looking, revealing a flocculent interior by transmitted light. Bottom colonies are thin, opalescent, and about 2 mm. in diameter. In slants the growth flows down, filling the V to a depth of 1 cm. or more, and leaving only a thin layer of growth on the slant. The mass of growth in the V is as deep in color as growth on beef-infusion agar. Large lenticular, granular bodies, looking like giant buried colonies, appear in the surface layer at the base of the slant in about two weeks.

BEEF BROTH.—Clouding is slow and moderate, beginning in about 48 hours. A pale-yellow rim begins to form in patches by the sixth day and is complete by the seventh or eighth day. Occasionally in undisturbed cultures a delicate incomplete pellicle is formed.

POTATO.—On potato cylinders growth is moderate, mustard yellow, smooth, glistening, and spreading, but not covering the entire surface of the potato. The water remains clear, with a moderate precipitate. The potato is grayed.

SYNTHETIC MEDIA.—Cohn's solution takes on a slightly milky appearance, but shows no rolling clouds on shaking. In Uschinsky's solution clouding is weak after 10 days' growth and only moderate, with a small amount of precipitate and with neither rim nor pellicle, at the end of two weeks. Moderate clouding occurs in Fermi's solution.

PHYSIOLOGY

LIQUEFACTION OF GELATIN.—Gelatin is very slowly liquefied at 18° to 21° C. In one test the surface growth after four days was about 4 mm. wide, lying in a dry depression 3 mm. deep. Two weeks later the gelatin was liquefied to a depth of 8 mm. and by the end of the fourth week the stratiform liquefaction was 12 mm. deep. The fluid was clear, with a heavy layer of yellow precipitate on top of the solid portion. No further liquefaction took place during the following month. In other tests evaporation kept pace with liquefaction so that only a dry pit was formed.

RELATION TO FREE OXYGEN.—The organism is aerobic. It does not grow in the lower end of stabs in beef agar or Thaxter's agar or in the lower half of shake agar cultures. No clouding occurs in the closed end of fermentation tubes.

FERMENTATION OF SUGARS.—Tests were made on beef-extract agar slants with bromcresol purple as indicator and 1 per cent, respectively, of the following carbohydrates: Sucrose, dextrose, galactose, levulose, maltose, lactose, glycerin, and mannit. The slants were streaked from agar cultures. In sucrose and levulose, acid was evident at the upper end of the slant after 24 hours. Two days later the whole of the slanted part of the culture was acid in sucrose, levulose, dextrose, and galactose. In maltose the slant was acid on the tenth day. On the fifteenth day the medium was acid throughout in all of these sugars except maltose, which never became as acid as the others. An alkaline reaction started on the seventeenth day and 10 days later the medium was alkaline throughout. In lactose, glycerin, and mannit there was a very slight color change, indicating an acid reaction, after two weeks' growth. Phenol red was then used as an indicator in the same kind of agar with each of these three carbohydrates. Acid was evident on the fourth day in glycerin and mannit, and on the tenth day the whole of the medium was acid. The lactose cultures began to show acid on the tenth day and were acid throughout on the seventeenth day. In fermentation tubes containing these carbohydrates in peptone water, the closed end does not cloud and no gas is formed.

REACTION IN MILK.—There is no evidence of growth in milk for several days. By the fifth day a yellow rim and surface layer of yellow growth 3 mm. deep has formed. These gradually increase to a depth of 1 cm. The milk remains fluid for about a month, although a soft curd may form during this period. In older cultures a solid curd forms with more or less separation of a clear yellow whey. Reduction of litmus is slow, beginning about the eighth day throughout the medium, and is complete in three weeks. No acid reaction appears before reduction has taken place, but the medium turns red when the cultures are 6 to 8 weeks

old. Reduction of methylene blue proceeds from the bottom upward, beginning on the third day, and is complete in 7 to 10 days. No crystals are formed.

HYDROLYSIS OF STARCH.—On starch-agar plates streaks which had made good growth were flooded with iodine when 7 days old. This produced a line 12 mm. wide around the growth, varying from deep purple in the outer portion to pale purple in the inner part nearer the growth, and in some cases colorless in the 1 mm. adjacent to the growth.

REDUCTION OF NITRATES.—Nitrates are not reduced. Tests were made in nitrate-bouillon cultures with the starch potassium iodide-sulphuric acid test and with the α-naphthylamine-sulphanilic acid test, and in the series of special media recommended in the Manual of Methods (*27*) for organisms giving a negative reaction in the simpler tests.

OPTIMUM pH FOR GROWTH.—In a series of broths made with beef infusion and Bacto-peptone, ranging from pH 4.4 to 9.6, growth was evident on the second day at pH 6.1 to 8.3 and was best at pH 7.7 to 8.3. By the fifth day there was clouding at pH 9.0 (at this time the checks had a pH value of 8.6), but there was no growth in the acid range beyond pH 6.1. By the seventh day a rim had formed in the culture at pH 7.5 to 8.2, but not in the others. Berridge (*1*) records growth with this organism at pH 5.0 to 9.2 in peptone broth made with Jardox. On agar slants growth was decidedly heavier at pH 7.6 and 7.9 than at pH 6.9. On agar plates 2-day-old colonies were barely visible at pH 7.0, whereas they were 1 mm. wide at pH 7.9.

TOLERATION OF SODIUM CHLORIDE.—The organism is sensitive to sodium chloride. Growth is moderate in beef broth at pH 7.0 containing 1 per cent Na Cl, weak in broth containing 2 per cent, and does not occur in broth containing 3 per cent.

OTHER PHYSIOLOGICAL REACTIONS.—*Aplanobacter michiganense* does not form indol or hydrogen sulphide. There is slight ammonia production in beef-peptone media, as shown on sugar agars containing bromcresol purple and by strips of filter paper dipped in Nessler's solution and inserted in beef-broth cultures.

TEMPERATURE RELATIONS.—The thermal death point ranges from 50° to 53° C. with different isolations. Tests were made with beef-infusion broth at pH 7.8, inoculated from 24-hour-old beef-broth cultures that had been heavily enough inoculated from an agar slant to cloud well in that time. Smith (*25*) reported no growth after exposure at 47°. The best growth is made at 25° to 27°. There is weak growth at 33° and moderate growth at 29°. No growth occurs in beef broth at 35° or 37°, but cultures were not killed by exposure for 10 days at these temperatures, as there was prompt clouding on removal to room temperature. Very slow clouding occurs at 1°. The organism has withstood year-round outdoor temperatures in Washington, D.C., and Geneva, N.Y.,[8] in sealed test tubes of sterilized soil, both moist and dry.

RESISTANCE TO DESICCATION.—*Aplanobacter michiganense* has survived eight months' drying on seeds and on cover glasses [9] and 2½ years in dry, sterilized soil without complete loss of virulence.

QUICK DIAGNOSIS

Slow-growing yellow colonies from typical lesions appearing on the third day on beef-agar plates, made up of small nonmotile, Gram-positive rods, may be safely diagnosed as *Aplanobacter michiganense.*

BRIEF CHARACTERIZATION

Aplanobacter michiganense is a short, nonmotile, Gram-positive, nonacid-fast rod occurring singly or in pairs, 0.6 to 0.7 μ wide by 0.7 to 1.2 μ long. It forms round, opaque, mustard-yellow colonies on beef agar; clouds beef broth slowly and forms a pale-yellow rim; has very weak diastatic action; liquefies gelatin very slowly; forms acid without gas from dextrose, sucrose, galactose, levulose, and maltose, and a very slight acid from lactose, glycerin, and mannit; does not reduce nitrates; causes very slow coagulation of milk without peptonization; reduces litmus and methylene blue in milk but does not form tyrosine crystals; produces ammonia but no indol or hydrogen sulphide; is aerobic; grows best at pH 7.5 to 8.2; withstands drying for 8 months on cover glasses and 2½ years in soil; optimum temperature is between 25° and 27° C.; thermal death point, 53°. Pathogenic to tomatoes.

[8] This result was obtained through the cooperation of J. G. Horsfall of the Geneva station.

[9] Tests on seed and cover glasses were not carried beyond 8 months, but the organism has been isolated from the interior of 2½-year-old seed.

SUMMARY

Bacterial canker has become more widespread and destructive in important tomato-growing sections in recent years. It causes the death of the plants at any stage from seedling to mature plant with a set of fruit. Losses range from a trace to 100 per cent. The disease is characterized by stunting of the plants; a gradual wilting and irregular dying of the leaves; streaking and cracking open of the stems, petioles, and veins; and the final collapse of the whole plant. The bacteria enter the fruit and penetrate the seeds through the vascular system.

During wet seasons bacterial canker also causes a very disfiguring fruit spot which renders the fruit unmarketable but does not cause it to rot. The spots are small but numerous and are characterized by noticeable white halos. Aside from this fruit spot there is no serious field spread.

The causal bacteria are disseminated on and in the seed. External disinfection should be practiced. Hot-water treatment has been found impracticable under commercial conditions, since temperatures that kill the bacteria are too close to the point at which injury to the seed occurs. According to present information, absolutely clean seed can be obtained only by selecting seed from fields free from this disease. Seed-bed sanitation is important, since the percentage of infection from infected seed is in part determined by seed-bed conditions that cause spread from plant to plant. These conditions are under investigation. Rotation of crops should be practiced, since it has been demonstrated that the bacteria are able to overwinter in the soil in some parts of the country.

The cultural and physiological characteristics of *Aplanobacter michiganense*, the causal organism, are given. Two variants, one an albino, are described.

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